Methods: Five hospitals in New York City contributed prospectively defined data elements on 10,847 patients undergoing percutaneous coronary intervention between January 1, 1998 and October 31, 1999. We analyzed the outcomes of 1112 patients from this database who underwent PPTCA for AMI with or without GPI treatment.
Results: Of the 1112 patients undergoing PPTCA for AMI, 336 (30%) were treated with GPI. GPI-treated patients were younger (61 years vs. 63 years, P=0.02), more often male (77% vs. 70%, P=0.02)and less often hypertensive (57% vs. 66%, P=0.003)compared to patients not treated with GPI. There were no significant differences with regard to diabetes, congestive heart failure, previous MI or prior bypass surgery between GPI-treated and untreated patients. Mean ejection fraction (49% vs. 47%) did not differ between groups. Hemodynamic instability was more common in the GPI-treated patients (17% vs. 9%, P<0.001). Patients treated with GPI were less likely to receive a stent (78% vs. 88%, P<0.001) than untreated patients. Procedural success was 98% in the GPI group and 97% for untreated patients (P=NS). The rates of post-procedure stroke, abrupt closure and stent thrombosis were equal in both groups. Pre-procedure CK levels were similar in both groups (681 U/L vs. 663 U/L, P=NS). The proportion of GPI-treated and untreated patients with CK levels >5 times normal at 8 (40% vs. 45%, P=NS) and 24 hours (37% vs. 34%, P=NS) were also similar. In-hospital mortality was 3% in the GPI-treated group compared to 2.2% in the untreated group (P=NS). After adjustment for differences in baseline characteristics, the risk of in-hospital mortality remained unchanged by GPI treatment.
Conclusion: GPI do not appear to have an effect on in-hospital outcomes following PPTCA for AMI.

POSTER SESSION

1124 Acute Coronary Syndromes: Risk Markers and Clinical Outcomes

Monday, March 18, 2002, 3:00 p.m.-5:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: 4:00 p.m.-5:00 p.m.

1124-27 TENAX-XR a:SiC-H Coated Stent Versus Noncoated Stents in 485 Patients With Acute Coronary Syndrome: 9- and 18-Month Follow-Up: The TRUST Trial

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The TENAX-XR stent is a stainless steel stent covered by a thin layer of silicon carbide by plasma enhanced chemical vapour deposition. This coating markedly reduces cellular aggregation. TRUST is a multi-centre randomized trial investigating the efficacy of this device in Unstable Angina Braunwald IIb-IIIb patients. All patients were clinically assessed upon discharge, 1, 3, 6, 9 and 18 months. All patients received the usual premedication, in addition Aspirin and Clopidogrel (or Ticlopidin) was given for 30 days. GPIIb / IIIa antagonists were given at the discretion of the operator, in only 2.1%. Succesrate for the TENAX-XR was 91%, in the reference group 87%. There were no deaths during the initial hospitalization; one myocardial Infarction occurred in the reference group, one PCI was required in each of the two groups. The primary endpoint, combination of a death, AMI and ischemia indicated target lesion revascularisation was virtually identical at 6 and 9 month in the Braunwald IIb group (9.6 %). However, in the Braunwald IIIb group there was a significant difference: TENAX-XR at 4.7 % vs 15.3 % in the reference group (p=0.02) with this tendency continuing at 9 month and 18 month follow-up.
Conclusion. Early PCI (0.4 ± 1.1 day after admission for severe symptoms) can be carried out at an extremely low risk with standard anti-thrombotic therapy including ASA and Clopidogrel. IIb/IIIa GP inhibitors apparently are not required. These data support the experimental data obtained with the silicon carbide coating. They suggest that with the initial 30 day treatment of ASA + Clopidogrel, the continued beneficial action of the coating leads to a marked reduction of MACE at 6, 9 and 18 months.

1124-28 807CC Genotype of the Platelet Gp Ia Could Represent a Protective Factor for Recurrences of Acute Coronary Events in Acute Coronary Syndromes

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Introduction: Glycoprotein Ia/IIa is an heterodimeric membrane complex that mediates platelet adesion to collagen, inducing platelets to aggregate. Recently a functional genetic polymorphism of the Ia subunit has been reported, consisting in a C807T transition (linked with A873G), associated with a variable expression of the platelet surface receptor. The lower receptor density of 807CC genotype may predispose to a hypocoagulable state and, then, to a reduction of the recurrence risk of Acute Coronary Events (ACE) in Acute coronary Syndromes (ACS). Methods: we enrolled 88 patients (pts), 61 with Acute Myocardial Infarction (AMI) and 27 with severe Unstable Angina (UA) as the first manifestation of disease occuring before 65 years of age, following them until 120 months. Results: 40 pts (23 AMI, 17 UA; 7 females, 52 ± 3 years) showed the 807 CC genotype, 38 pts (28 AMI, 10 UA; 7 females, mean age 53 years) the 807CT genotype, 10 pts (10 AMI; all males, mean age 54) the 807TT genotype. Pts with 807CC genotype showed a free-event median survival significantly longer than pts with 807CT or 807 TT genotype either in the entire group (64 months vs 44 months; p<0.05; risk ratio=0.4889 95%CI 0.25-0.99) and especially in the 61 pts with AMI. (p<0.03; risk ratio=0.35, 95%CI 0.1817-0.9159). Finally no statistically significant difference was found between 807CT and 807 CT genotypes. Conclusions: these data suggest that in the ACS, pts with 807CC genotype, characterized by a lower collagen receptor density on platelet membrane, could have a lower recurrence risk of ACE compared to 807T allele carriers.

1124-29 Heparin/PF4 Complex Antibodies in Patients With Unstable Angina: Evaluation of Cardiac Thrombotic Events in 24 Months of Follow-Up

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Background. Unfractionated heparin (UH) and low molecular weight heparin (LMWH) play an important role in standard therapy of unstable angina. The treatment with UH can induce in a variable number of patients antibodies against heparin/PF4 complex. The aim of the present study is to evaluate cardiac events in 24 months follow-up in patients (pts) treated with UH or LMWH.
Methods. We evaluated 114 pts (71 men, of mean age 68 ± 11 years, range 46-85 years) admitted to our facility with unstable angina. They were randomized in two groups: group A treated with UH and group B treated with LMWH. An immunoassay ELISA test was used to detect anti-heparin/PF4 antibodies. Blood samples were taken at baseline and on the 7th and the 40th day. The mean follow-up period was of 21.5 ± 15.2 months, (group B 23.2 ± 17.8 months and group A 19.68 ± 14.8 months). End-points were: acute myocardial infarction, angina, need for revascularization, stroke and death for all causes. Statistical analysis included a Kaplan and Maier analysis and the Log Rank Test.
Results. The frequency of a positive ELISA did not vary with patient's age or gender. In group A 55% pts were positive for heparin/PF4 antibodies at ELISA on day 40. In group B none pts tested positive for heparin/PF4 antibodies. Group A was divided in 2 subgroups according to presence (group AAc+) or absence (group AAc-) of antibodies. We reported a significant difference in cumulative events between group B and group AAc+ (Log rank=7,57; p=0,005 Fig.1). No difference was observed between group B and group AAc- (Log rank=0.22; p=0,6382).
Conclusions. Pts treated with LMWH had a better prognosis compared with pts treated with UH. We observed that the difference exists only between pts that developed antibodies, suggesting a link between anti-heparin/PF4 antibodies and thrombotic cardiac events.

1124-30 Risk of Ischemic Events Can Be Predicted by Angioscopic Evaluation of Yellow Plaques

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Background: Elevated C-reactive protein (CRP) has been proved a marker of increased risk of atherothrombotic clinical events. Disruption of lipid-rich plaque, which is detected by angiscopy as yellow plaque, is regarded as one of the major mechanisms of acute coronary syndrome and atherosclerosis progression. Therefore, we examined the relation between ischemic events after PCI with CRP elevation or with progression of yellow plaque formation.
Methods: Angioscopic evaluation was performed in 131 patients when they received PCI. The number of yellow plaques in a coronary artery was counted by angioscopy. Serum concentrations of CRP (N < 0.2 mg/l) were measured on admission. They were followed up for new ischemic events, i.e., acute myocardial infarction (AMI) and unstable or stable angina, for 4.9±0.8 years. The recurrence of angina due to restenosis was not counted as new ischemic event.
Results: New ischemic events were documented in 19 patients (AMI occurred in 4 patients, and effort angina in 15 patients). Although there was no relationship (p=0.101) between serum CRP and the occurrence of ischemic events, the patients who suffered new ischemic event had more yellow plaques (2.5±1.3 vs. 1.9±0.9; p=0.049) than those who did not. The incidence of AMI (13.1% vs. 0.14%, p=0.015) and of new ischemic events (39.1% vs. 13.9%, p=0.008) were significantly higher in the patients who had >2 yellow plaques than those who had ≤2 yellow plaques in the coronary artery that had culprit lesion.
Conclusions: The risk of suffering new ischemic events during 5-years after PCI can be evaluated by counting the number of yellow plaques in a coronary artery by angioscopy.

1124-45 Acute Myocardial Infarction in HIV-Infected Patients

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Background: Recent reported series of patients suggested that HIV-infected patients are at an increased risk of premature coronary artery disease. However, the mechanisms are not fully understood and there is little information regarding the clinical outcome of acute coronary syndrome in these patients.
Methods: We compared the characteristics and long-term course of 24 consecutive HIV-infected patients with AMI, and 48 age- and gender- matched non-HIV patients.
Results: The mean age was 47±49 and 88% were males in both groups. Twenty-two HIV-infected patients (92%) were receiving an anti-retroviral drug, 17 (71%) protease inhibitors (PI), and 13 (54%) statins. HIV-infected patients had lower cholesterol (175±49 vs 210±48, p=0.013), LDL (104±40 vs 136±44, p=0.011) and HDL values (32±9.7 vs 40±9.9, p=0.005) Otherwise there were no significant differences in risk factors for CAD. Angiographically, there were no significant differences in the incidence of multi-vessel CAD (76% vs 54%, p=0.15), and revascularization (83% vs 94%, p=0.31). Both HIV-infected and control patients had a relatively benign in-hospital course with no deaths or reinfarctions. There was also no significant difference in the indices of myocardial damage (peak troponin, LVEF). In a mean follow-up of 15 months, HIV-infected patients had higher incidence of re-infarction (20% vs 4%, p=0.07), recurrent coronary event (45% vs 11%, p=0.007), and recurrent acute atherothrombotic event (cardiovascular death, reinfarction, acute cerebro- or peripheral vascular event 30% vs 4.4%, p=0.008). Among patients who underwent PTCA, HIV-infected patients had higher incidence of target vessel revascularization (TVR) (43% vs 10.5%, p=0.016). TVR was particularly evident in-patients with increased viral load, irrespective of PI therapy. In HIV-infected patients, treatment with PI was not associated with higher cholesterol, LDL, or triglyceride levels,